COMPARATIVE ANALYSIS OF CLINICAL AND RADIOLOGICAL PARAMETERS OF INTRABONY DEFECTS TREATED WITH AUTOGENOUS BONE GRAFT AND XENOGRAFT COMBINED WITH PRP –

A 6 MONTHS STUDY

Dissertation submitted to

THE TAMILNADU Dr. M.G.R. MEDICAL UNIVERSITY

In partial fulfillment for the Degree of

MASTER OF DENTAL SURGERY

BRANCH II

PERIODONTICS

APRIL 2011

CERTIFICATE

This is to certify that ***Dr. T.ARTHIIE,*** Postgraduate student in the Department of Periodontics, J.K.K.Nattraja Dental College and Hospital, Komarapalyam has done this dissertation titled **"COMPARATIVE ANALYSIS OF CLINICAL AND RADIOLOGICAL PARAMETERS OF INTRABONY DEFECTS TREATED WITH AUTOGENOUS BONE GRAFTS AND XENOGRAFTS COMBINED WITH PRP- 6 MONTHS FOLLOW UP STUDY"** under my direct guidance during her post graduate study period 2008 -2011.

This dissertation is submitted to **THE TAMILNADU Dr. MGR MEDICAL UNIVERSITY** in partial fulfillment of the degree of **MASTER OF DENTAL SURGREY, BRANCH II – Periodontics.**

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ACKNOWLEDGEMENT

I am greatly indebted to ***Dr. Sugumari Elavarasu****, my Professor and Head, Department of Periodontics, J.K.K.Nattraja Dental College and Hospital, Komarapalayam, for her strenuous and dedicated efforts towards the post graduate students and for her invaluable guidance, support and encouragement throughout my post graduate study.*

I would like to extend my heartfelt gratitude to Professor ***Dr.S.Sivakumar****, Principal, J.K.K.Nattraja Dental College and Hospital, for his kindness in allowing me to utilize the facilities in the college.*

I take this opportunity to express my humble gratitude to ***Dr.Kermani,*** *Reader, for his guidance and support.*

I am highly obliged to my sir ***Dr.S.Thangakumaran****, Senior lecturer for his valuable suggestions, enthusiastic support and constant encouragement throughout my study.*

I sincerely thank ***Dr. S. Jaikarthik****, Senior lecturer who inspired me in every phase of my professional life. His profound knowledge, patience and perseverance and his incessant encouragement, guidance and support had benefited me and my colleagues in every facet of our academic life.*

My heartfelt gratitude to ***Dr. G.S. Jayaprakash****, Senior lecturer without whose guidance, constant support and encouragement, this dissertation work couldn't have been shaped to its present form.*

I sincerely thank ***Dr. Thangam****,* ***Mr.Siril*** *for their generous support rendered throughout my course.*

My heartful thanks to all my dear colleague's, and all the non-teaching staff's for their kind help during my postgraduate period.

I would like to thank all of my patients for their kind cooperation.

My sincere gratitude to my father ***Mr. G.P.Thangavelu****, mother* ***Mrs. M. Baggialakshmi****, and husband* ***Mr.V. Nandhivarman*** *without whom I would not have been able to reach this height.*

Finally, without the grace of the ALMIGHTY this possibility would have been just impossible.

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INTRODUCTION

Periodontitis is an inflammatory disease of supporting tissues of the teeth caused by specific microorganisms resulting in the progressive destruction of periodontal ligament, alveolar bone with pocket formation, and recession or both[1]

The purpose of periodontal therapy is to eliminate the inflammation of the periodontal tissues and to regenerate the periodontal attachment apparatus including cementum, functionally oriented periodontal ligament and alveolar bone.[2]

In general, Regeneration is defined as "Reproduction or reconstitution of a lost or injured tissue".[3] 'Periodontal regeneration' is defined histologically as "Regeneration of tooth's supporting structures including alveolar bone, periodontal ligament and cementum over a diseased root surface".[3] Thus the key to periodontal regeneration is to stimulate the progenitor cells to reoccupy the defects.[4]

Conventional periodontal treatments such as scaling and root planing are highly effective in repairing disease related defects and halting the progression of periodontitis. These treatments typically result in the development of long junctional epithelium between the root surface and gingival connective tissue rather than regrowth of tissue that restores the architecture and function.[5]

For decades, a number of surgical procedures have been advocated which includes open flap debridement, open flap debridement with bone grafts or bone substitutes and guided tissue regeneration. Open flap debridement may result in the formation of long junctional epithelium which is more susceptible to microbial invasion and is thought to be less stable attachment. Thus bone grafting is the most common form of regenerative therapy.[3,6] Autogenous bone grafts, bone derivatives (Allogenic grafts, Xenogenic grafts) and bone substitutes (Alloplastic materials) have

been used for periodontal regeneration. The use of these bone grafts results in the regrowth of alveolar bone and formation of new attachment which would be stimulated either by osteogenesis, osteoconduction, or osteoinduction.[7]

Among all these biomaterials autogenous bone grafts have been adopted as gold standard since there is possibility to retain cell viability, graft revascularization and no possibility of disease transmission.[6] Also they were considered to yield high osteogenic potential and used with the intent to improve the outcomes of periodontal regeneration.[5] These bone grafts contain live osteoblasts and osteoprogenitor cells and heal by osteogenesis.[7, 8]

Autogenous bone grafts can be harvested either from intraoral or extraoral donor sites. Multiple intraoral locations have been used to harvest bone grafts, including the maxillary tuberosity, exostoses, extraction sites and edentulous ridges. Another important source of intraoral autogenous bone grafts includes the harvesting of osseous coagulum, bone blend generated from osteoplasty or ostectomy.[8, 9]

The osseous coagulum is obtained by mixing the bone dust and blood. This produces small particle size which induces more bone formation and provides additional surface area for the interaction of vascular and cellular elements.[10] Recently, the autogenous bone scraper which is used in periodontics produces osseous coagulum of thin curled bone strips.[10,11] This scraper is more advantageous compared to other intraoral harvesting methods as it eliminates the need for second surgical site, and reduced postoperative pain and swelling and thus ultimately improves patient morbidity.[12]

The major disadvantages of autografts are donor site morbidity, procurement techniques, handling and processing of the harvested material, which have led to the introduction of allografts.[13] Allografts also may result in the transmission of pathogens. Their high cost and the shortage of supply of donor bone resulted in the introduction of Xenografts.[13] Xenograft is prepared by protein extraction of bovine bone that results in trabecular structure of hydroxy apatite similar to human cancellous bone. It enhances the rate of bone formation by osteoinduction, and seems to be compatible since its use has not been associated with any immunological reaction. [7, 14]

Allografts, Xenografts, Alloplasts donot possess inherent osteogenic properties and act only as a substrate for cell migration and proliferation and this led to the application of various biologically active substances such as peptide sequences, protein preparations, and polypeptide growth factors to enhance regeneration in both bone and periodontal defects.[15] Polypeptide growth factors are biologic mediators that regulate cellular events including cell proliferation, chemotaxsis, differentiation and matrix synthesis via binding to specific surface receptors.[15, 16]

Growth factors may be preserved in platelets. Platelets contain factors such as Platelet deriver growth factor (PDGF), Transforming growth factor- β (TGF-β), Vascular endothelial growth factor (VEGF), Insulin growth factor (IGF), Hepatocyte growth factor, Epidermal growth factor and these factors are involved in angiogenesis and osteogenesis.[16] Amongst these growth factors PDGF and TGF- β have been extensively studied and are known to be abundant in the alpha granules of platelets.[17]

A convenient approach to obtain autologous PDGF and TGF - β is the use of autologous platelet concentrate also known as Platelet Rich Plasma (PRP). PRP is a highly concentrated form of autogenous platelets and works via the degranulation, providing a rich and readily obtainable source of diverse group of growth factors.[17,18] These growth factors are concentrated to about 300 times than that of level normally present in plasma. [18] The use of PRP is based on its potential to release multiple wound healing growth factors and cytokines, which are responsible for increasing cell mitosis, increasing collagen production, recruiting other cells to the site of injury, initiating vascular in-growth and inducing cell differentiation.[19]

PRP stimulates fibroblastic and osteoblastic proliferation and suppresses epithelial cell proliferation.[18,20] PRP is believed to result in early consolidation and graft mineralization to increase the rate of bone formation and also promote 15–30 % increase in trabecular bone density.[21] Thus PRP could be an attractive and potent material for bone graft procedures as it has the ability to form hydrogel, suitable for cellular migration, and proliferation. Also the local concentration of secreted growth factors in PRP enhances the initial wound healing.[21]

Thus in the present study a combination of PRP and Xenograft is compared with Autogenous bone graft with respect to clinical and radiological findings after a period of six months in periodontal intrabony defects.

AIMS & OBJECTIVES

The aim of the present study is to compare Autogenous bone graft (Group 1) and Xenograft combined with PRP (Group 2). The clinical and radiological parameters were evaluated after a period of six months in periodontal intrabony defects.

The parameters for assessing the effectiveness of grafts are

1. Evaluation of change in probing depth following therapy.
2. Estimation of change in clinical attachment level following therapy.
3. Evaluation of amount of bone fill following therapy.

REVIEW OF LITERATURE

Periodontal regeneration :

The goal of tissue engineering and regenerative therapy is to promote healing and regeneration of tissue's structure and function.[22] Regenerative treatment modalities includes the use of three – dimensional biomaterial scaffolds or matrices to support the regeneration of tissues lost due to diseases. Thus the important goal of periodontal therapy is to obtain a reduced pocket depth after treatment in order to arrest further disease progression.[23, 24]

Cell types and molecules participating in periodontal regeneration :

1. *Cells*

a. Epithelial – Junctional epithelium.

b. Fibroblasts – Gingival fibroblasts, Periodontal ligament fibroblasts.

c. Bone cells – Osteoblasts, Osteoclasts, Osteocytes.

d. Cementoblasts.

2. Molecules

a. Growth factors – Fibroblast growth factors (FGF) 1and 2, Insulin like growth (IGF) factors I and II, BMP's, Epidermal growth factor (EGF), Platelet derived growth factor (PDGF)

b. Adhesion molecules – Fibronectin, laminin, Osteopontin, collagens.

c. Structural proteins – Type I, III, V, XII, XIV Collagens.[19]

Melcher A.H (1976) stated that regeneration of the periodontal ligament is the key to new attachment because, it provides continuity between the alveolar bone and cementum and contains cells that can synthesize and remodel the three connective tissues of the periodontium.[3] He also stated that the types of cell which repopulate the root surface after periodontal surgery determines the nature of attachment that would eventually form. After periodontal flap surgery, the debrided root surface may be repopulated by four different types of cell: Epithelial cells, Cells derived from the gingival connective tissue. (gingival fibroblasts), Cells derived from the bone (osteoblasts,osteoclasts), Cells derived from the periodontal ligament (periodontal ligament fibroblasts).[3]

Osseous defects :

Osseous defects can be classified into infrabony and suprabony defects. An infrabony defect is a type of osseous defect in which its base is apical to the crestal margin of the alveolar bone. Vertical defects or angular defects are those that occur in an oblique direction leaving a hollowed out trough in the bone along the side of the root.[1]

Goldman HM and Cohen DW (1958)[25] classified angular defects on the basis of the number of walls:

· **Three wall defect :**

One in which the defect has three bony walls and the tooth constitutes the fourth wall.

· **Two wall defect :**

One in which the defect is delineated by two bony walls and a root surface.

· **One wall defect :**

One in which only one bony wall and the root surface remain.

Glickman I and Carranza FA added one more type of angular defect to ***Goldman and Cohen's*** classification and called it the **combined osseous defect:** where the number of walls in the apical portion of the defect could be greater than that in its occlusal portion.[1] If an infrabony defect is present, there is a net loss of the attachment apparatus ie; bone, periodontal ligament and cementum. Intrabony defects, especially the three-wall defect, provide the best opportunity for regaining the lost periodontal attachment by using bone substitutes.[1, 5]

Bone grafts :

Bone grafting is usually done to establish a new attachment apparatus at a more coronal level than that existing in a particular diseased tissue.[26] Grafting biomaterials include autogenous grafts, allogenic grafts, xenogenic grafts and alloplastic materials. The assumption behind the clinical use of grafting procedures is that the complete regeneration of the attachment apparatus including new bone formation and new connective tissue attachment would be enhanced by the various biomaterials due to their **osteogenetic** potential, **osteoinductive** capability, or **osteoconductive** properties.[7] Osteogenesis refers to the formation or development of new bone cells contained in the graft. Osteoinduction is a chemical process by which molecules contained in the graft convert the neighboring cells into osteoblasts.

Osteoconduction is a physical effect by which the matrix of the graft forms a scaffold that favours outside cells to penetrate the graft and form new bone.[26]

Bowers GM, Chadroff B, and Carneval R (1989)[24] evaluated the new attachment apparatus formation in humans and the value of graft materials in enhancing the formation of new bone, cementum, and periodontal ligament. Biopsies were obtained at 6-months and evaluated. Results indicated that a significantly more new attachment apparatus, new cementum and new bone formed in grafted than nongrafted sites. They concluded that the combination of highly osteogenic materials and epithelial exclusion techniques offer promise for enhancing the amount, frequency and predictability of periodontal regeneration.

Aichelmann-Reidy ME, Yukna RA et al. (1998)[26] reviewed about the various bone substitutes in the treatment of intrabony defects. The results showed that significant reduction in clinical probing depth and improved gain in attachment levels compared to flap debridement surgery alone for periodontal osseous defects.

Reynolds MA, Elizabeth M (2003)[8] reviewed the efficacy of bone replacement grafts in the treatment of periodontal osseous defects. The results showed that bone grafts increases bone fill, reduces crestal bone loss, increases clinical attachment level and reduces probing depth compared to open flap debridement. Histologically they concluded that new attachment apparatus was observed in intrabony defects following bone grafting where as open flap debridement resulted in repair characterized by the formation of long junctional epithelium.

Autogenous bone grafts:

The use of bone grafts for reconstructing osseous defects produced by periodontal disease dates back to ***Hegedus in 1923*** and was revieved by ***Nabers & O'Leary in 1965***.[27] Multiple grafting materials have been used to clinically improve, via regeneration, the prognosis of teeth within intrabony defects. Among them, autogenous bone is regarded as the gold standard for bone regeneration, due to its intrinsic characteristics that provide optimal conditions for angiogenesis and migration of cells with osteogenic potential. The autogenous bone graft in contrast to both allografts and xenografts has osseoinductive, osseoconductive properties and is immunologic free.[6]

Sources of Autografts:

The only materials with human histological evidence to substantiate their regenerative use are autogenous bone grafts that can be harvested from either intraoral or extraoral donor sites. Multiple intraoral locations have been used to harvest bone grafts, including the maxillary tuberosity, exostoses, healing wounds and extraction sites and edentulous ridges. Another important source of graft material includes the harvesting of osseous coagulum, bone blend generated from osteoplasty or ostectomy.[7,8] ***Robinson E*** first described the technique of osseous coagulum by mixing the bone dust and blood. This produces small particle size which induces more bone formation and provides additional surface area for the interaction of vascular and cellular elements.[10]

Thus many investigators have reported on the clinically successful use of autogenous bone grafts harvested from intraoral sites in the treatment of intrabony defects. It yields regenerative responses superior to those obtained following surgical debridement procedures alone.[8] Moreover, long-term evaluations suggest that the regenerative gain achieved by autogenous bone grafts remains clinically stable.[28]

Robinson E et al. (1968)[10] conducted an in vivo study to evaluate the potential of Osseous coagulum for bone induction. He used high speed turbines in conjunction with carbide burs to obtain osseous coagulum. The patient had a 7 mm pocket on the mesial aspect of a mandibular second premolar and osseous coagulum was grafted. At three months pocket depth reduced to 2-3 mm. Reentry procedures after 3, 4, 6, 12, 18 and 24 months showed considerable amount of bone fill. They also concluded that the smaller the particle size of the donor bone, the more certain are its resorption and replacement.

Schallhorn RG, and Denv MS (1968)[29] studied the use of autogenous hip marrow biopsy implants for bony crater defects. They selected patients with moderately advanced periodontitis localized or multiple interproximal bony crater defects of 4 to 5 mm apical to the crests of the facial and lingual bony plates. On reflaping the areas, it was apparent that the defects were completely filled with osseous tissue. Thus they concluded that autogenous bone can be successfully used for the eradication of interproximal bony crater defects.

Robinson E (1969)[30] used osseous coagulum for bone induction in which they mixed autogenous cortical bone chips with blood and placed in periodontal defects. He concluded that smaller particle size of the donor bone leads to rapid resorption and replacement of host bone and thus osteogenesis occurs at a rapid rate.

Halliday DG et al. (1969)[31] grafted newly formed autogenous bone in the osseous defects. The donor site he preferred was edentulous area of the mandible. He then treated mandibular first molar with pocket depth of 7 mm in 3 patients with intraoral cancellous autogenous bonegrafts. He concluded that in all 3 cases, the bone in the area of reattachment is more radiolucent than the surrounding bone and showed complete success in attaining reattachment after 9 months.

Rivault A, Toto P, Garguilo A et al (1971)[32] studied the histologic healing phenomena in periodontal defects corrected by the osseous coagulum procedure in 4 adult rhesus monkey. The defects between first and second premolars and second premolar and first molar were grafted with intraoral autografts. They concluded that osteogenic stimulus which induce the undifferentiated mesenchymal cells to become osteoblasts, originates in the osseous walls of the defects as well as on the graft material. The graft material undergoes necrosis and its components appear to be used for the build up of new bone.

Haggerty PC, Maeda I et al. (1971)[9] treated 10 vertical bone defects with autogenous bone grafts. They concluded that autogenous bone grafts can be used successfully in the surgical armamentarium to restore the attachment apparatus around periodontally diseased teeth.

Hiatt W, Schallhorn RG, and Aaronaian RJ (1972)[33] transplanted intraoral cancellous bone and marrow which was taken from maxillary tuberosity area into 166 periodontal osseous defects in 40 patients. They concluded that an average bone fill of 3.44 mm was obtained. The greatest fill came from those defects with great number of bony walls. The cancellous bone has considerable surface area and more number of osteoclasts when compared to iliac marrow. There is no evidence of root resorption while using intraoral autografts.

Coverly L, Toto P, and Garguilo A (1975)[34] histologically evaluated the regeneration of osseous coagulum in surgically created 2 and 3 wall defect of 4 young adult female rhesus monkey and grafted those sites with osseous coagulum. They concluded that the use of osseous coagulum led to a more rapid osteogenesis compared to correction by curettage alone.

Forum SJ, Thaler R, Scopp IW et al. (1975)[35] studied about the histological responses of osseous coagulum in 3 patients with infrabony defects. Histologic findings were obtained from the grafted site 6-13 weeks postoperatively and concluded that remodeling involved the osseous walls, periodontal ligament (PDL), cementum and graft spicules. Also there is marked increase in cementogenesis at the grafted sites.

Hawley CE, Miller J (1975)[36] showed that 28 months following free osseous autograft therapy, microscopically the periodontal structures were reconstructed with new bone, PDL and cementum and also found that osteogenesis occurred at a rapid rate.

Carraro JJ, Sznajder N and Alonso CA (1976)[2] treated 56 infrabony pockets with one wall and two wall bony defects with intraoral cancellous bone and 44 defects were treated with open curettage without bonegrafts. They concluded that more favourable results (new attachment) were obtained in treatment of infrabony pockets when intraoral cancellous bone is used in association with conventional techniques of flap surgery compared to open curettage alone. Also observed that, in 2 wall bony defects the use of bonegrafts results in a greater area of new attachment.

Hiatt W, Schallhorn RG, Aaromain RJ et al. (1976)[37] studied the microscopic examination of human bone marrow allograft and autograft in 100 human block section.They concluded that both allografts and autografts yielded new attachment apparatus including new cementum, bone and functionally oriented PDL. Also no ankylosis or root resorption was noted in fresh intraoral donor material and with frozen iliac autografts.

Mellonig JT (1991)[14] reviewed the treatment of bone autografts and allografts in periodontal therapy. The various graft materials were discussed with respect to case reports, controlled clinical trials, and human histology. The study concluded that autogenous bone grafts can be used successfully in periodontal therapy. Also multiple histologic reports suggest that regeneration of a new attachment apparatus is possible with different types of autogenous bone grafts. Root resorption and ankylosis may be observed only following grafts of fresh iliac cancellous bone and marrow.

Becker W, Burton et al. (1998)[38] compared Demineralized Freeze-Dried Bone Allografts (DFDBA) and autogenous bone in human extraction sockets. 7 patients were selected and biopsy done between 3-13 months. The study concluded

that DFDBA sites revealed the presence of dead particles with no evidence of osteoclastic resorption and appear to delay normal bone formation. Autologous sites revealed vascular channels with woven lamellar bone and were undergoing active osteoclastic resorption.

Erpenstein H, Diedrich P et al. (2001)[39] evaluated the performance of two bone mills (R Quetin Bone Mill and Micro Knochenmühle, Aesculap) for the grinding of autogenous bone (intraoral, cortical) according to the following criteria: (1) loss of bone during the grinding process, (2) particle size of the chips, (3) variability in chip size, (4) technical handling, and (5) cost-benefit ratio. The size and variability of the bone particles were determined histomorphometrically. They concluded that, to promote osteogenetic activity, small particles of graft were considered as the best. The Quetin mill was superior in all points to the Aesculap mill for the requirements of a periodontal practice.

Cochran DL, Jones A, Heijl L et al (2003)[40] evaluated the periodontal regeneration with a combination of enamel matrix proteins and autogenous bone grafts. Periodontal defects ranging in size from 1-6 mm were selected. The results showed that enamel matrix proteins plus autogenous bone grafts treatment resulted in greater tissue formation than controls. They concluded that enamel matrix proteins and autogenous bone represents a therapeutic approach that can be highly effective in stimulating significant amounts of periodontal regeneration.

Choi CS, Chai CS, and Wikesijo (2005)[6] evaluated the periodontal healing with focus on root resorption and ankylosis following implantation with autogenous bone and coral derived biomaterial in intrabony defects in dogs. After 8 weeks

histological analysis done and concluded that particulated autogenous bone and coral derived biomaterial may be implanted into periodontal defects without significant healing observation such as root resorption and ankylosis.

Orsini M, Orsini G et al. (2008) [41] studied the long term clinical results on the use of autogenous bone grafts in the treatment of intrabony defects. They compared the use of autografts with calcium sulfate and autografts covered with membrane.12 subjects were selected in the spilt mouth trial. The results showed that, at 6 months there was a probing depth reduction and improved clinical attachment level in both the groups. Thus they concluded that both therapies led to significant short and long term improvement in the outcome variables and autogenous bone grafts appears to have a valuable role in treating periodontal defects.

Abolfazli N, Saber FS, Lafzi A, Eskandari A et al. (2008)[11] conducted a study to compare cerabone (A Decalcified Freeze-dried Bone Allograft) with Autogenous Bone Graft (ABG) in the treatment of two and three-wall intrabony periodontal defects. A total of 5 patients with 10 pairs of intrabony defects received surgical therapy. 10 sites were treated with DFDBA and 10 sites were treated with ABG. After 6 months bone fill and defect resolution significantly improved in both groups. The study concluded that both graft materials were beneficial for treatment of intrabony defects.

Sangeetha singh (2010)[42] conducted a study to evaluate the regenerative potential of intra-oral autogenous bone grafts in the treatment of intrabony defects in patients with generalized aggressive periodontitis. The study concluded that

autogenous bone grafts produced a significant probing depth reduction and bone fill at 6 months.

Bone substitutes:

Bone substitutes will play a pivotal role in the future of periodontal regeneration. They are synthetically derived or processed from exoskeletons of other species (xenograft) and are an alternative to autogenous or allogeneic bone replacement grafts.[7] The main concerns over autogenous graft are related to donor site morbidity and graft resorption, and these have triggered the development of new conceptual grafting approaches in order to provide efficient alternatives to autogenous bone, either of fill bone gaps or to attain large bone augmentations.[8] Also the quality of autogenous bone is variable depending on the health status of the patients who are in greatest need of the best material to promote regeneration. Even in healthy patients, the disadvantages of a limited supply, increased procedure time and post operative pain and risk of surgical complications at the harvest sites led to the substantial effort to develop an off-the-shelf autograft substitute.[14] These includes allogenic, xenogenic, synthetic graft materials which function primarily by passively guiding or conducting cell migration through the matrix, eventually leading to repair of the defect.[26]

Osteoinduction is the formation of new bone by inducing the differentiation of undifferentiated mesenchymal cells through stimuli provided by the demineralized bone matrix. The process of osteoinduction requires the presence of a collagen or proteoglycan matrix and bioactive proteins such as Bone Morphogenic protein (BMP).[43] Osteoinductive potential of commercially available DFDBA is subject to the bioavailability of BMP in its active form. BMP has been shown to up regulate the

expression of cbfa-1128 -the master switch that regulates osteoblast differentiation. BMP exerts its effects primarily through the Smad pathway although other mechanisms have been suggested. A truly inductive material must be capable of supporting the differentiation of uncommitted bone marrow stromal cells to osteoblast for optimal bone regeneration in periodontal defects.[14, 44]

Bowers GM, Granet M, Stevens M (1985) [45] evaluated the potential for regeneration of a new attachment in patients whose attachment apparatus had been destroyed by periodontal disease. Debrided intrabony defects were treated with and without demineralized freeze-dried bone allograft. Biopsies were obtained in 6 months and regeneration was evaluated histometrically. Preliminary results in 7 patients and 24 intrabony defects indicate that new attachment was observed when intrabony defects were grafted with demineralized freeze-dried bone allograft. New attachment was not observed in nongrafted sites.

Sonis ST, Williams RC, Marjorie K et al. (1985)[46] Studied histologically, clinically and radiographically to evaluate the sequence of healing following implantation of bovine demineralized bone powder (DBP) into severe, spontaneous periodontal defects in beagle dogs. No evidence of localized inflammatory response or delayed hypersensitivity reaction was noted. Histologic evaluation demonstrated the presence of DBP at 1 month following implantation, but the material was replaced with new bone by 3rd month. An intact epithelial attachment appeared at the 1st month after the implantation of DBP. They concluded that DBP did not appear to predispose to external root resorption and successfully induced new bone formation.

Reynolds MA and Bowers GM (1996) [5] observed histologically the fate of DFDBA in human intrabony defects. Histologic sections showed defects harboring residual graft particles exhibited significantly greater amounts of new attachment apparatus formation including new bone, cementum, and associated periodontal ligament than sites without evidence of graft matrix.

Fucini SE, Quintero G et al. (1993)[47] compared the bony defect resolution obtained using two different particle size ranges of DFDBA. Paired interproximal intrabony periodontal defects in 11 patients were grafted with DFDBA. Soft and hard tissue measurements were made using an electronic constant-force probe at the initial and reentry surgeries. Treated sites in 10 patients were reevaluated by reentry approximately 6 months postoperatively. Mean bony defect fill was 1.66 mm for the large particle group and 1.32 mm for the small particle group. There was no statistically significant difference in bony fill between defects grafted with the different particle sizes of DFDBA when used in humans.

Brunsvold MA, Mellonig JT et al (2000)[48] did a study to evaluate bovine derived xenograft in the treatment of human periodontal defects. 4 patients with radiographic evidence of vertical bone loss were selected and grafted with bovine derived xenograft. After 6 months block sections were taken. Histologically they found the formation of new bone, cementum, and PDL, coronal to the base of the reference point. Thus the study concluded that periodontal regeneration is possible following grafting bovine derived xenograft.

Growth factors:

The lack of a predictable outcome when using passive therapies, such as osteoconductive matrices and guided tissue regeneration, led to the development of treatments designed to stimulate the cells responsible for regeneration.[19] The tissue engineering combines three key elements to enhance regeneration: conductive scaffolds, signaling molecules and cells.[19] The important biological event involved in tissue regeneration is specific cell directed migration.[19] A variety of naturally occurring potent bioactive proteins are known to be present in bone, platelets, and a number of other cells and tissues and these regulates events in tissue engineering.[49]

Polypeptide growth factors (PGF) are naturally occurring biological modifiers that have the potential to alter the host tissue to stimulate or regulate the wound healing process. They regulate key cellular events in tissue regeneration, including cell proliferation, chemotaxsis, differentiation, and matrix synthesis via binding to specific cell surface receptors.[49] Growth factors either singly or in combination have been used and experimental evidence for bone regeneration has been documented in both animal and human trials.[50]

Terranova VP and Wikesjo (1987)[51] reviewed extracellular matrices and polypeptide growth factors as mediators of functions of cells of the periodontium. The polypeptide growth factors are hormone-like in both structure and function. Factors which modulate cell chemotaxis have recently been implicated in cellular growth and differentiation. In addition, endothelial cell growth factor has been shown to be a potent chemoattractant for human endothelial cells while platelet-derived growth factor has been shown to be a chemoattractant for smooth muscle cells and

fibroblasts. Thus, the study concluded that both PGFs and isolated components of the extracellular matrix appear to play an increasingly important role in our understanding of tissue definition.

Committee on Research, Science and Therapy of American Academy of Periodontology (1996)[52] is intended for members of the dental profession. They reviewed the various aspects of Growth factors (GFs) on cells and tissues involved in periodontal wound healing. According to them growth factors are a class of biological mediators involved in repair and regeneration, regulates several key cellular processes such as mitogenesis, chemotaxis, differentiation, and metabolism. The sequence of events necessary for periodontal regeneration relies on the above processes for osteogenesis, cementogenesis, and connective tissue formation. They concluded that numerous preclinical in vitro and in vivo studies have demonstrated that certain growth factors modulate putative components of periodontal wound healing resulting in substantial regeneration of the periodontium in animals.

Platelet Rich Plasma:

Platelets are a rich source of naturally occurring growth factors, which can play an important role in regeneration of periodontal tissue. PRP is procured from whole blood and is rich in platelets and naturally occurring autologous growth factors that are present in plasma.[17,53] Its use is based on the potential of the plasma to release multiple wound-healing growth factors and cytokines, which are responsible for increasing cell mitosis, increasing collagen production, recruiting other cells to the site of injury, initiating vascular in-growth and inducing cell differentiation.[57] Released growth factors such as PDGF, TGF-β, IGF and EGF have been shown to

have an osteoregenerative potential because of their pro-angiogenic effects and differentiating effects on osteoblasts. Recently, a combination of PRP and bone graft has been advocated as a means of increasing the rate of osteogenesis and enhancing bone formation qualitatively. Moreover, it has been suggested that PRP may promote a 15% to 30% increase in the trabecular bone density.[15]

Contents of PRP:

The large numbers of platelets found in PRP release significant quantities of mitogenic polypeptides, such as PDGF, TGF-β, as well as IGF-I.[17]

Platelet derived growth factor:

Platelet derived growth factor is a glycoprotein with a molecular mass of approximately 30kDa. It is the primary growth factor in platelets, and also synthesized and secreted by other cells such as macrophages and endothelial cells.[16] The PDGF family consists of four members, PDGF-A, PDGF-B and newly identified PDGF-C and PDGF-D, which form four functional homodimers, PDGF-AA, PDGF-BB, PDGF-CC, PDGF-DD as well as heterodimer PDGF-AB, as endogenous cell products. PDGF-A, PDGF-B chains are expressed on most cell types such as fibroblasts, osteoblasts, endothelial cells and macrophages, and PDGF-C, PDGF-D are expressd in various tissues. There are approximately 0.06 ng of PDGF per 1 million platelets. This calculates to 6×10gram of PDGF, or about 1200 molecules of PDGF, per individual platelet.[17, 53]

It stimulates chemotaxis of fibroblasts, neutrophils and macrophages. Activates macrophages, induce proliferation of fibroblasts and stimulates the production of the extracellular matrix components, fibronectin.[54] PDGF is responsible for mitogenesis, causing an increase in number of healing cells, angiogenesis, generating development of new capillaries and up regulation of other growth factors and cells resulting in promotion of fibroblastic and osteoblastic functions, promotion of cellular differentiation, and acceleration of the effects of growth factors on other cells.[17]

Transforming Growth Factor –β:

Transforming growth factor-β is a term applied to a super family of growth factors and differentiating factors of which the bone morphogenetic proteins (BMPs) are members. The three isoforms of TGF-b (b1, b2 and b3) have a broad range of activity within healing. TGF-b1 is the most abundant in all tissues and is the form found in platelets.[17] It is chemotactic for monocytes, lymphocytes, and fibroblasts. It stimulates endothelial cell proliferation and tubule formation. TGF-β plays a central role in regulating maturation and strength of wounds.[55]

Insulin like growth factors:

Insulin-like Growth Factor-I (IGF-I) and Insulin-like Growth Factor-II (IGF-II) are usually thought of as growth factors secreted by osteoblasts during bone formation to increase numbers of osteoblasts and thereby accelerate bone deposition. Both IGF-I and IGF-II are relatively small proteins with molecular masses of 7.7kd and 7.5kd, respectively. They bind to a specific IGF cell membrane receptor that excites kinase activity (formation of a high-energy phosphate bond) to a cytoplasmic

signal protein. IGFs are released to couple new bone formation to bone resorption. The presence of IGF in platelets would be expected to act on precursors of osteoblasts i.e., those cells already committed to an osteoblast lineage and on endosteal osteoblasts, which are the cells that produce the initial phase I bone in bone grafts. Insulin-like growth factors are therefore mitogenic to osteoblast lineage cells and are also stimulators of bone formation from existing differentiated osteoblasts.[17]

Platelet derived epidermal growth factor (PDEGF):

PDEGF was discovered by Cohen in 1962 and was the first growth factor described. It stimulates epidermal regeneration, promotes wound healing by stimulating the proliferation of keratinocytes and dermal fibroblasts and enhances the effects and production of other growth factors.[17,56]

Platelet derived angiogenesis factor (PDAF):

PDAF has the capacity to induce vascularization in vivo. It stimulates vascular endothelial cells and is involved in the process by which new blood vessels invade devascularized tissue. [17, 56]

Platelet factor-4 (PF-4):

PF – 4 is a chemoattractant for neutrophils released from alpha granules, which may be partially responsible for the influx of neutrophils into wounds. It acts as chemoattractant for fibroblast and is a potent anti-heparin agent.[17]

Harvesting or Procurement of PRP:

Several recent studies have highlighted different methods to harvest PRP and its numerous advantages I the healing of bone, bone grafts and soft tissue.

The Automated Systems:

A number of methods make the use of electronic gradient density centrifugation for separation of blood and plasma.

Marx et al (1998)[17] in a study obtained PRP by means of an electromedics gradient density cell separator. This cell separator withdraws 400-450 ml of venous blood via venous catheter. With a centrifuge speed of 5600 rpm, blood is with drawn at a rate of 50ml/min as the separator adds citrate phosphate dextrose at 1:5 ml ratio. Blood is centrifuged into three basic components i.e. red cells, PRP (buffy coat) and Platelet poor plasma (PPP). Once PPP is collected, the centrifuge speed is reduced to 2400 rpm to allow for precise separation of PRP and PPP. To initiate the coagulation process PRP is mixed with 10% calcium chloride and 10,000 units of bovine thrombin. The resultant gel was mixed with bone graft and packed into the recipient site. Platelets counts showed an increase of 338% in PRP compared to baseline count.

Juan Obarrio and Jose Dutare (2000)[15] in a case report on growth factors in periodontal surgery, used the ELMD-500 medtronics transfusion system for procuring PRP.

The Test tube or Manual isolation techniques (Vacuatainer system):

The test tube method contained a lower concentration of platelets compared to the automated system, it was the most economical and contains adequate amount of growth factors namely, PDGF-AB, PDGF-BB, TGF-β.

Sonnleitner and Sullivan (2000)[58] presented a simplified technique for producing PRP for intraoral grafting techniques using The Test tube or Manual isolation techniques (Vacuatainer system). 6ml of blood was collected in a test tube containing anticoagulant and centrifuged for 20 minutes at 1200 rpm. This results in two layers i.e, lower opaque red cell fraction also containing WBCs and Platelets and a second upper straw yellow turbid fraction called the serum component. Then the entire serum component was pipetted out. Test tube was centrifuged again for 15 minutes at 2000 rpm. This resulted in the formation of a platelet concentration (PRP) and the resulting supernatant PPP. The top 80% of PPP was pipetted out. The residual PRP was mixed with 0.1 ml of thrombin and 10% calcium chloride to procure the gel.

The test tube method has also been advocated by ***Landesberg[59] and Glickman 2000, Aghaloo and Freymiller 2002, Kim and Park 2002, Weibrich G 2002.***

Thus the incorporation of platelet rich plasma (PRP) with bone graft produces dense, vital bone in a shorter interval. The growth factors has been shown to accelerate bone repair, promote fibroblast proliferation, increase tissue vascularity, rate of collagen formation and mitosis of mesenchymal stem cells and endothelial cells, as well as osteoblasts, thus playing key roles in the rate and extent of bone formation.[54]

Matsuda N, ***Cho I, Genco RJ et al. (1992)***[60] did an in vitro study to evaluate the mitogenic, chemotactic and synthetic responses of rat PDL fibroblastic cells to EGF, TGF -β, rh PDGF-AB, rh PDGF-BB, natural (n) PDGF –AB and IGF. The maximum mitogenic effect of PDGFs were observed at the concentrations at 10ng/ml, where as IGF-I was seen at concentration higher than 100ng/ml. TGF- β, showed inhibitory mitogenic responses. The study concluded that rh PDGF-BB, IGF-I stimulate proliferation and chemotaxsis of PDL fibroblastic cells. rh PDFG-AB also stimulates collagen synthesis by PDL fibroblastic cells. Thus rh PDGF-BB and IFG-1 may have important roles in promotion of PDL healing and may be useful for clinical application in periodontal regeneration.

Oates TW, Rouse CA, Cochran DL et al. (1993)[61] conducted in vitro study to determine the effects of PDGF on human PDL cells. The results of the study demonstrated that both PDGF AA & BB enhance the mitogenic activity in a dose dependent manner over a concentration range of 1-5 mg/ml. They concluded that PDGF – AA and BB are major mitogens for human PDL cells.

Marx RE, Carlson, Ralph M, and Eichstaedt (1998)[17] used autologous platelet concentrates to identify the growth factors present within them. The additional amount of these growth factors obtained by APC to grafts evidenced a radiographic maturation 1.62 to 2.16 times that of grafts without APC as assessed by histometry and radiographic evidence.

Mccauley LK, Somumam MJ et al. (1998)[16] studied about the biologic modifiers in periodontal regeneration. They suggested that many of the biologic modifiers mainly growth factors like PDFG, TGF- β, have significant influences on

cell behaviour and show great promise for use in regenerative therapies. Also they discussed that the additional investigations are required both at the molecular level and at clinical level to improve the predictability of regenerative therapies.

Moonti-Cho (1998)[63] studied the effect of platelet derived growth factor in guided tissue regenerative therapy. The lesions selected were protected by an expanded polytetrafluroethylene barrier membrane and PDGF - BB. The study concluded that the application of PDGF-BB showed potent chemotactic and mitogenic effects on PDL fibroblasts. The sustained release of PDGF – BB also contributes to the repopulation of fibroblast synthesis of extracellular matrix components in the wound.

De Obarrio JJ, Arauz-Dutari JI, Chamberlin TM et al. (2000) [15] conducted an in vitro study to demonstrate a new biotechnology in which platelet gel is used in combination with demineralized freeze dried bone allograft for treatment of periodontal osseous defects. 5 patients with serve localized bone loss were treated with PRP and DFDBA. Evaluation at 2^{nd} and 6^{th} months they found significant reduction in probing depth. Also the reentry at 2 years revealed significant bone fill of the periodontal osseous defects. New bone formation was evident and confirmed by IOPA. Thus they concluded that new biotechnology significantly enhanced periodontal regeneration and wound healing.

Shanaman R, Flistein MR, Danesh-Meyer MJ et al. (2001)[63] did a study to evaluate the potential of PRP in combination with bone allograft to enhance bone regeneration in alveolar ridge defects. Augmentation resulted in clinical and radiographic gain in both vertical and horizontal components of osseous defects. They

suggested that PRP used in conjugation with various bone derivatives can support new bone formation in localized alveolar ridge defects. PRP also improved the handling properties of the graft material with which it was combined, thereby facilitating graft placement and stability.

Lekovic, Camargo PM, Weinlaender M, Vasilic N et al. (2002)[64] conducted a study to compare the effects of PRP / BPBM / GTR and GTR alone. They selected 18 systemically healthy patients having two similar interproximal defects with pocket depths of 6 mm and defects were surgically treated with an absorbable membrane GTR or a combination of PRP / BPBM / GTR. At 6 months pocket depth reduction was 4.98 for PRP / BPBM / GTR and 3.62 mm for GTR. The gain in clinical attachment observed was 4.37 mm for PRP / BPBM / GTR and 2.31 mm for GTR groups. PRP and BPBM provides an added regeneration effect to GTR in promoting the clinical attachment of intrabony defects.

Okuda K, ***Kawase T, Momose M et al. (2003)***[65] conducted in vitro study to demonstrate that PRP contains high levels of PDGF and TFG- β. Evaluation for PDGF – AB and TGF- β1 was done using ELISA kits. The results showed the levels of PDGF-AB and TGF- β1 were 182 ng/ml and 140 ng/ml respectively. The study concluded that PRP can serve as a source for the GFs such as PDGF and TGF- β. In addition PRP also modulates cell proliferation and suppresses epithelial cell proliferation. The suppression of the downgrowth of junctional epithelium onto the dental root surfaces in the regeneration process would avoid interference by epithelium with the formation of new connective tissue attachment on the root surface and thus clinical application of PRP act as a potent tool to facilitate periodontal regeneration.

Hanna R, Trejo M and Weltman RL (2004)[20] conducted randomized, spilt mouth clinical trial to compare the clinical outcomes obtained by the combination of PRP and Bovine derived xenograft (BDX) to those obtained from the use of the bone replacement graft alone in the treatment of intrabony defects. 13 patients with attachment loss of ≥ 6 mm were selected. At 6 months probing depth reduction of 3.54 mm and 2.53 mm obtained in BDX with PRP and BDX alone respectively. The study concluded that addition of high concentration of autologous platelets to BDX to treat intrabony defects significantly improved their clinical periodontal response.

Quyang Xiang Ying, Qiao Jing et al. (2006)[66] evaluated the effectiveness of PRP as an adjunct to bovine porous bone mineral (BPBM) graft in the treatment of human intrabony defects. 10 patients with intrabony defects > 6mm were selected. 5 defects were treated with BPBM alone in split mouth design. After 1 year Probing depth reduction was 4.78mm in BPBM / PRP and 3.48 mm in BPBM alone. CAL gain is about 4.52mm, 2.85 mm in BPBM /PRP and BPBM respectively. The study concluded that additional biological effects of PRP may contribute to the improvement of clinical outcome in the combined group (PRP+BPBM). The secreted GFs immediately bind to their transmembrane receptors on adult mesenchymal stem cells, osteoblasts, fibroblasts, endothelial cells and cause cellular proliferation, matrix formation, osteoid production and collagen synthesis. Thus a combination of PRP and BPBM led to us significantly more favourable clinical improvement in periodontal intrabony defects compared to BPBM alone.

Torres J, Tamimi F, Tresguerres IF et al. (2009)[67] conducted a study to evaluate the combination of anorganic bovine bone graft (ABB) with PRP. 16 healthy 6 months old female rabbits were used and created calvarial defects were grafted with

ABB or PRP or ABB with PRP. 6 weeks after augmentation, mixture of PRP with ABB produced twice the vertical bone volume of ABB alone. Thus the study conducted that the combination of anorganic bovine bone and PRP resulted in increased vertical bone augmentation when compared with autologous blood in similarly sized created defects.

Ogino Y, Ayukawa Y, Kukita T et al. (2009)[68] conducted an in vivo study to evaluate the effects of PRP and PPP on osteoclastogenesis with rat bone marrow cell culture. The results showed that PRP decreased the number of TRAP positive multinucleated cells in a dose-dependent manner and also the amount of osteoprotegerin produced from rat bone marrow cells. They concluded that that PRP suppresses osteoclastogenesis, therefore inhibiting bone resorption. In addition they also demonstrated that PRP increased the secretion of osteoprotegerin, an inhibitor for osteoclast formation.

Creeper F, Lichanska AM, Marshall RI et al (2009)[69] aimed to investigate the in vitro effect of PRP on osteoblasts and periodontal ligament cell function. PRP is used to deliver growth factors, in a safe and convenient manner for enhancing bone and periodontal regeneration. The results showed that PRP and PPP had stimulatory effects on the migration and cellular proliferation of both human osteoblasts and periodontal ligament cells. They concluded that PRP can exert a positive effect on osteoblast and periodontal ligament cell function. PPP also appears to have the ability to promote wound healing-associated function.

Nagata, Melo (2009)[70] histologically analysed the effect of autogenous platelet-rich plasma (PRP), on healing of autogenous bone (AB) grafts placed in surgically created critical-size defects in rabbit. The results showed that AB and AB/PRP significantly improved bone formation and a beneficial effect of PRP was limited to an initial healing period of 4 weeks.

Kotsovilis S, Markou N, Pepelassi E et al. (2010)[71] conducted randomized controlled clinical trials (RCTs) to evaluate the adjunctive use of PRP in the therapy of periodontal intraosseous defects. Data sources were obtained from electronic databases, manually searched journals and contact with experts. They concluded that the clinical use of PRP is an entirely safe procedure, causing no adverse events or postoperative complications. Diverse outcomes (positive and negative) have been reported for the efficacy of PRP combined with various therapeutic bioactive agents or procedures, reflecting the limited and heterogeneous data available and possibly suggested that the specific selection of agents or procedures combined with PRP could be important. Additional research on the efficacy of each specific combination of PRP is necessary.

Materials And Methods

A randomized, split mouth, single evaluator; 6 months prospective clinical study was conducted to evaluate the clinical and radiographic parameters in periodontal intrabony defects using autografts and xenografts with PRP. Prior to initiating the study the patients were informed of the purpose and design of this clinical trial and were requested to sign an informed consent for the study. The ethical clearance was obtained from the institutional ethical board. Patients were selected from out patient Department of Periodontics, J.K.K. Nattaraja dental college and Hospitals, Komarapalayam using the following selection criteria.

Inclusion criteria:

1. Age limit of 20-50 years of both sexes.

2. Probing depth $\geq$ 5mm as assessed by William's graduated periodontal probe.

3. Patients with a minimum of two contralateral intrabony defects.

4. Vital teeth.

Exclusion criteria:

1. Known systemic diseases, short and long term drug therapies.

2. Drug allergies.

3. Pregnant and lactating women.

4. Teeth with traumatic occlusion.

6. Smokers.

STUDY DESIGN:

A split mouth design was followed, where two sites in the contra lateral quadrants with probing pocket depth of ≥ 5mm with radiographic evidence of bone loss at baseline were chosen. Probing pocket depth standardization was done with acrylic stent in all the selected areas.

CRITERIA FOR GROUPING

Group1: 7 intrabony defects treated with autogenous bone grafts.

Group2: 7 intrabony defects treated with xenogenic grafts and PRP.

CLINICAL PARAMETERS

The following variables were measured at baseline, 3 months & 6 months post surgery.

1. Gingival index
2. Plaque index
3. Oral hygiene index (simplified)
4. Probing pocket depth - deepest probing depth was measured.
5. Clinical attachment level.

1) Gingival Index: (Loe. H and Silness. P, 1963)

The soft tissue surrounding each tooth were divided into 4 gingival scoring units i.e. the distofacial papilla, the facial margin, the mesiofacial papilla and the entire lingual margin. A periodontal probe was used to assess the bleeding of the gingival tissues on probing.

Gingival units were assessed according to the following criteria:

0 - Normal gingiva

1 - Mild inflammation, slight change in color, slight edema, no bleeding on palpation.

2 - Moderate inflammation, redness, edema & glazing, bleeding on probing.

3 -Severe inflammation, marked redness & edema, ulceration, tendency for spontaneous bleeding.

The gingival index score for each of the 4 gingival surfaces was given a score from 0 to 3. The scores around each tooth were totaled and divided by four and the gingival index score for each tooth was obtained.

The scoring criteria are as follows

0.1 – 1.0 -Mild

1.1 – 2.0 -Moderate.

2.1 - 3.0 - Severe.

2) Plaque index: (Silness. P and Loe. H, 1964)

0 - No plaque in the gingival area.

1 - A film of plaque adhering to the free gingival margin and adjacent area of the tooth. The plaque may be recognized only by running a probe across the tooth surface.

2 - Moderate accumulation of soft deposits within the gingival pocket & on the gingival margin or adjacent tooth surface that can be seen by the naked eye.

3 - Abundance of soft matter within the gingival pocket or on the gingival margin & adjacent tooth surface.

The areas examined were distofacial, facial, mesiofacial and lingual surface, using explorer. The plaque score was obtained by totaling the four plaque scores per tooth and then divided by four. The plaque score per person is obtained by adding the plaque score per tooth and dividing by the number of teeth examined.

The scoring criteria are as follows

0.1 -1.7 - Good.

1.8 - 3.4 - Fair.

3.5 – 5.0 - Poor.

3) Oral hygiene index (Green & Vermillion 1964):

The six tooth surfaces were examined. Facial surface of 16, 11, 26, 31 and lingual surface of 36, 46 were examined using an explorer.

Debris index (DI - S)

Dental explorer was placed on the incisal third of the tooth and moved towards the gingival third of the tooth.

The scoring criteria is

0 - No debris or stain present.

1 -Soft debris covering not more than 1/3rd of the tooth surface or the presence of extrinsic stains without other debris, regardless of surface area covered.

2 -Soft debris, covering more than 1/3rd but not more than 2/3rd of exposed tooth surface.

3 -Soft debris covering more than 2/3rd of the exposed tooth surface.

DI - S score per person is obtained by totaling the debris score per tooth surface & divided by number of surfaces examined.

Calculus index: (CI – S)

Assessed by placing a dental explorer into the distal gingival crevice and drawing it sublingually from distal contact area to the mesial contact area.

0 – No calculus present.

1– Supragingival calculus covering more than 1/3rd of the exposed tooth surface.

2-Supragingival calculus covering more than 1/3rd but not more than 2/3rd of the exposed tooth surface, or the presence of the individual flecks of subgingival calculus around the cervical portion of the tooth or both.

3- Supragingival calculus covering more than 2/3rd of the exposed tooth surface or a continuous heavy band of subgingival calculus around the cervical portion of the tooth or both.

The clinical level of DI, CI are

Good - 0.0 - 0.6

Fair - 0.7 - 1.8

Poor - 1.9 – 3.0

The OHI-S score per person is the total of DI-S and CI -S scores per person.

The scoring criteria are as follows

0.0 - 1.2 - Good

1.3 – 3.0 - Fair

3.1 – 6.0 - Poor

4) Probing pocket depth:

The depth of the pocket was measured at selected sites using William's graduated periodontal probe. The probe was inserted parallel to the long axis of the tooth gently, until resistance was felt and the readings were recorded to the nearest millimeter from the gingival margin to the base of the pocket. Acrylic stents were used to standardize the path of insertion and angulation of the probe.

5) Clinical attachment level:

The level of attachment is the distance between the base of the pocket and Cementoenamel Junction (CEJ) or a fixed point. The distance from the CEJ (if CEJ is not detected, the coronal border of the stent was used) to the base of the pocket was measured. The readings were recorded to the nearest millimeter.

Occlusal stents for positioning and measuring probes were fabricated with cold cured acrylic resin on a cast model obtained from an alginate impression. Notch was made on the stent to permit and standardize the entry of periodontal probe into the pocket. The occlusal stent was made to cover the occlusal surfaces of the tooth being treated and occlusal surface of one tooth in the mesial and distal directions. The stents were also extended apically on the buccal and lingual surfaces to cover the coronal third of teeth involved. [66, 72]

RADIOGRAPHIC PARAMETERS:

An intraoral periapical radiograph (IOPA) of each defect site was exposed. Exposure was made at 226 volts at 0.6 second and Kodak E – speed plus films were used. The film to object and focal spot to object distances were standardized to 20cm. Digitized images were displayed on the monitor at 5X magnification using Adobe Photoshop 7.0 computer software. A 0.5mm grid was made on the digitized images and all linear measurements were made using Auto-CAD 2006 software.[73]

PRESURGICAL THERAPY:

For all the enrolled patients routine blood investigations were taken. The initial therapy consisted of oral hygiene instructions, scaling & root planing. Three weeks following phase I therapy, a periodontal re-evaluation was performed. The study used a split mouth design where two interproximal sites were assigned to PRP and Xenograft or Autogenous bone graft.

PRP preparation:

10ml of blood was drawn from patients by a venipuncture of the antecubital vein 30 minutes prior to surgery. Blood was transferred to a sterile glass tube containing the anti-coagulant (3.8% sodium citrate). The test tube was gently shaken so that blood and anticoagulant got thoroughly mixed. The initial centrifugation process was done at 1200 r.p.m for 15 minutes at room temperature.[66] This results in separation of 2 basic fractions,

1. Lower opaque red cell fractions containing White blood cells, Platelets and red cells.

2. Upper straw yellow turbid fraction which is the serum.

Serum is then pippetted out into new test tube and the remaining red cell fraction is centrifuged at 2000 rpm for 20 minutes. The second centrifugation results in

1. **Platelet poor plasma (PPP) on the top** of the preparation which contains few platelets.

2. **Middle layer comprising of PRP** which consists of platelets and White blood cells.

3. **Bottom most** fraction comprising of **red blood corpuscles** which also contains newly synthesized platelet at the top most layer.

80% of PPP was pipetted out and discarded. PRP was then pippetted along with some red blood cell fraction and collected in a separate sterile glass tube.[66]

SURGICAL PROCEDURES:

All the periodontal surgical procedures were performed on patients under aseptic conditions by a single operator following presurgical phase. The patient was anaesthetized using lignocaine 2% with 1:1,00,000 epinephrine as a vasoconstrictor. Using Bard – Parker blade number 15 buccal and lingual sulcular incisions were made to elevate the mucoperiosteal flaps. Pocket epithelium and granulation tissue from the inner surface of flaps were carefully removed. Thorough soft tissue debridement & root planing were accomplished with Hu - Friedy curettes. The surgical area was then rinsed with copious amounts of sterile saline.

Surgical procedure for Group 1:

With the autogenous bone scraper (Ebner grafter, Salvin Dental Specialists, USA) bone shavings were obtained from the site adjacent to the defect area. The curved blade shaves bone when raked with appropriate pressure and angled at 25-30°

from the bone surface with a pull stroke, produces very thin curled bone strips. These bone shavings were collected in a collection chamber. While scraping, shavings combine with blood and flow into collection chamber forming osseous coagulum. Later the collected autogenous bone graft was grafted in the intrabony defect.

Surgical procedure for Group 2:

PRP was mixed with 10% calcium chloride to facilitate coagulation and to activate platelets before application. Osseograft (Advanced Biotech Products (P) Ltd India) is a demineralized bone matrix composed of type I collagen derived from bovine cortical bone samples (Xenograft), and particles of approximately 250 micrometers. Osseograft was emptied into a sterile dappen dish and PRP was added until the mixture becomes applicable. Increments of the graft material were added, to the bottom of the defect, and were condensed with an amalgam condenser to adapt the particles to the defect until it was completely filled. After grafting, flaps were repositioned to accomplish as much as possible complete inter proximal closure. The flaps were approximated with simple interrupted sutures using 3-0 silk thread. Post surgical instructions (Appended) were given to the patient and recalled after one week for suture removal and follow up.

APPENDIX-1

Instructions to the Patient

1. Report immediately on developing any untoward reactions like pain, swelling, hypersensitivity, drug allergies.
2. Should report to the dental office if secondary bleeding persists within 24 hours.
3. Should avoid intake of any hot and hard foods.
4. Patient was advised to take antibiotic every 8 hours for 3 days and analgesic every 12 hours.
5. Avoid brushing the treated area for 1 week from the day of surgery; use cotton tip applicator (Johnson and Johnson ear buds) to gently clean the area.
6. Not to use dental floss and toothpicks at the site.
7. 0.12%chlorhexidine mouth rinse twice daily.
8. Follow up visits have to be done in 24 and 48 hours.
9. The patients were asked to perform regular oral hygiene habits by appropriate brushing technique using tooth brush and tooth paste.
10. The patients were instructed to report on the subsequent appointment.

APPENDIX-2

PROFORMA

PATIENT NAME**:** OP NO**:**

AGE: SEX:

ADDRESS: PHONE NO:

CHIEF COMPLAINT:

SITE SELECTED:

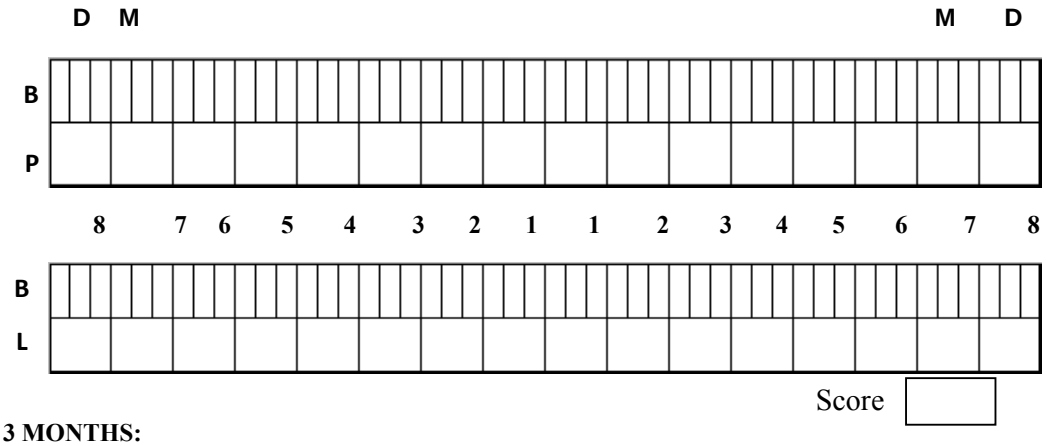
GROUP 1: (AUTOGRAFTS) ☐

GROUP 2: (XENOGRAFT+PRP) ☐

INDICES

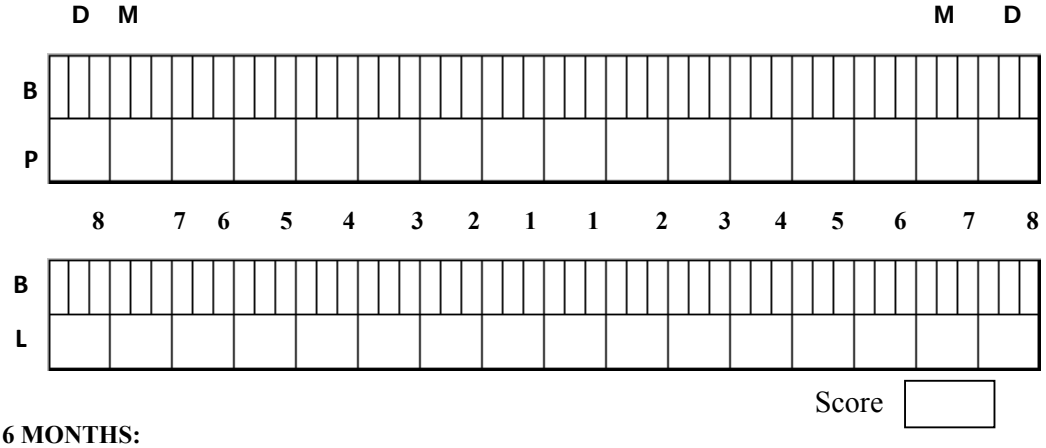
GINGIVAL INDEX

BASELINE:



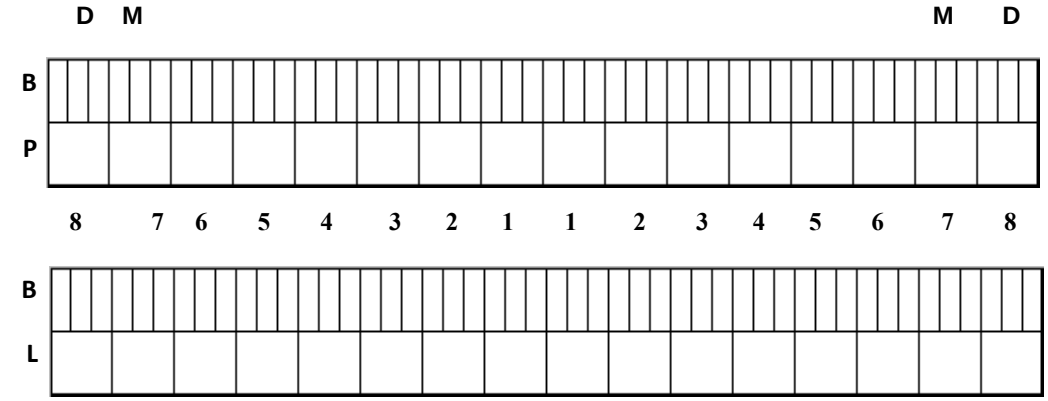
Score

3 MONTHS:



Score

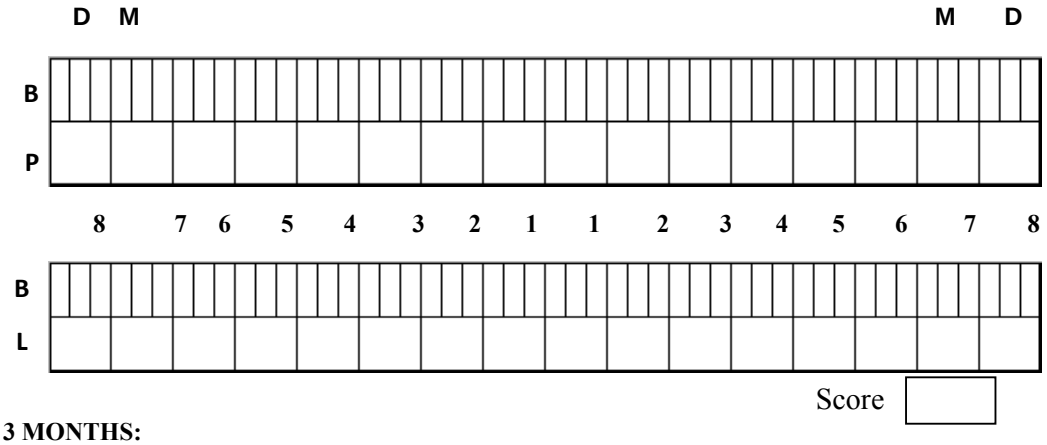
6 MONTHS:



Score

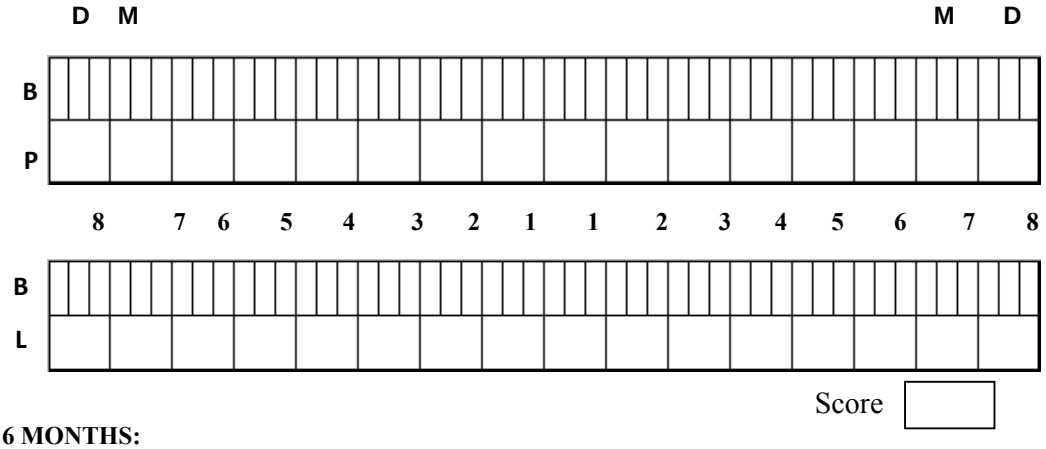
PLAQUE INDEX

BASELINE:



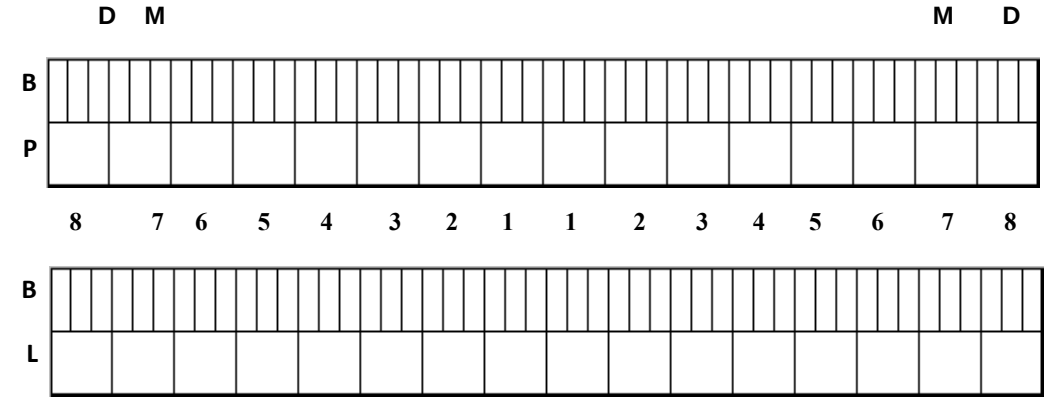
Score

3 MONTHS:



Score

6 MONTHS:



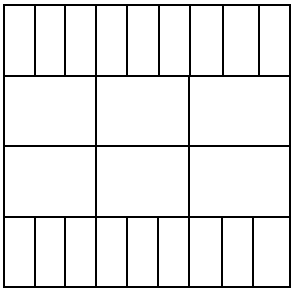
Score

ORAL HYGIENE INDEX

Debris index (DI)

BASELINE:

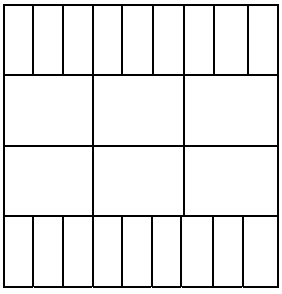
16 11 26



46 31 36 **SCORE** ☐

3RD MONTH:

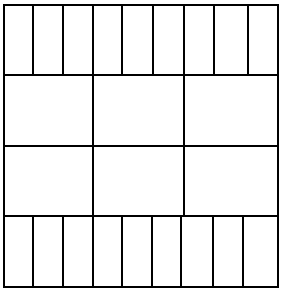
16 11 26



46 31 36 **SCORE** ☐

6TH MONTH:

16 11 26

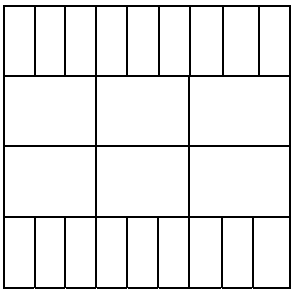


46 31 36 **SCORE** ☐

Calculus index (DI)

BASELINE:

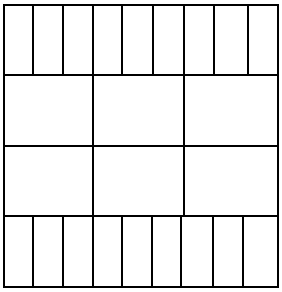
16 11 26



46 31 36 **SCORE**

3RD MONTH:

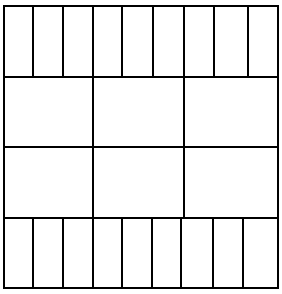
16 11 26



46 31 36 **SCORE**

6TH MONTH:

16 11 26



46 31 36 **SCORE**

OHI SCORE (DI+CI) =

INTERPRETATION:

CLINICAL PARAMETERS:

DATA	BASELINE		3 RD MONTH		6 TH MONTH	
	GROUP 1	GROUP 2	GROUP 1	GROUP 2	GROUP 1	GROUP 2
Probing pocket Depth (mm)						
Clinical Attachment Level (mm)						

RADIOGRAPHIC FINDINGS:

DATA	BASELINE		3 MONTHS		6 MONTHS	
	GROUP 1	GROUP 2	GROUP 1	GROUP 2	GROUP 1	GROUP 2
Bottom of the defect to CEJ						

INFORMED CONSENT OBTAINED FROM THE PATIENT

DEPARTMENT OF PERIODONTICS , J .K .K NATARAJA DENTAL COLLEGE , KOMARAPALAYAM, NAMAKKAL DISTRICT**.**

PATIENT NAME:

I have been explained about the nature and purpose of the study in which, I have been asked to participate. I understand that I am free to withdraw my consent and discontinue at any time without prejudice to me or effect on my treatment.

I have been given the opportunity to question about the material and study. I have also given the consent for photographs to be taken at the beginning, during and end of the study. I agree to participate in this study.

I hereby give the consent to be included in "COMPARITIVE ANALYSIS OF CLINICAL AND RADIOLOGICAL PARAMETERS OF INTRABONY DEFECTS TREATED WITH AUTOGRAFT AND XENOGRAFT COMBINED WITH PRP".

Station: **SIGNATURE OF PATIENT**

Date: **SIGNATURE OF PROFESSOR**

APPENDIX-3

ARMAMENTARIUM

MATERIALS AND INSTRUMENTS USED FOR PERIODONTAL FLAP SURGERY:

- Gloves
- Mouth mask
- Patient apron
- Chair apron
- Head cap
- Sterile cotton rolls
- Gauze
- Saline
- Kidney tray
- Betadine
- Lignocaine
- Syringe
- Mouth mirror
- Straight Probe
- Explorer
- William's graduated periodontal probe
- Tweezer
- Tissue holding forceps
- Bard-Parker handle
- Bard-Parker blade number:11, 15

- Periosteal elevator
- Hu-Friedy Gracey Curettes
- Ultrasonic scalers
- Needle holder
- 3-0 silk suture
- Scissors
- Dappendish
- Autogenous bone scraper (Ebner grafter - Salvin Dental Specialists, USA)
- Osseograft (Advanced Biotech Products (P) Ltd India)
- Plastic instrument
- Amalgam condenser.

MATERIALS AND INSTRUMENTS FOR PRP PREPARATION

- 10 ml Syringe
- Torniquet
- Pippettes
- Test tubes
- Centrifuge (R8C laboratory centrifuge, Remi equipments, Mumbai)
- Calcium chloride
- Anti-coagulant

RESULTS

In this study **Student *t* – distribution (**William Sealy Gosset) is used to analyze the significance between the groups at different time intervals.

The *t* –distribution is used when the sample size is small (less than 30) and standard deviation of the population is unknown.

Paired *t* -Test:

When there is a direct relationship between each specific data point in the first and second set, such as measurements on the same subject before and after the study, then the paired *t*-test will be appropriate.

According to this test,

The *t* – statistic is defined as

$$t = \frac{\overline{X}_1 - \overline{X}_2}{S} \times \sqrt{\frac{n_1 n_2}{n_1 + n_2}}$$

$\overline{X}_1$ - mean of the first sample.

$\overline{X}_2$ - mean of the second sample.

n_1 - number of observations in the first sample.

n_2 - number of observations in the second sample

S - combined standard deviation.

Thus

p<0.001 was considered as highly significant at 1% level of significance**.**

p>0.05 was considered as not significant at 5% level of significance.

Plaque index (PI):

The mean plaque index score at baseline was 1.14 ±0.68, at 3rd month was 0.80±0.52 and at 6th month was 0.51±0.42. The values at 3rd and 6th month were not statistically significant when compared to baseline; with a p-value >0.05 as shown in Table no.1 and Graph no.1.

Oral hygiene index (OHI):

The mean oral hygiene index score at baseline was 1.04±0.70, at 3rd month was 0.72±0.52 and at 6th month was 0.52±0.42. Comparing with the baseline value, it was not statistically significant at 3rd and 6th month time intervals with a p-value >0.05 as shown in Table no.1 and Graph no.1.

Gingival index:

At baseline the mean gingival index score was 1.02±0.53, reduced to 0.73±0.48, 0.42±0.32 at the end of 3 months and 6 months respectively. The values at 3rd and 6th month were not statistically significant when compared to baseline with a p-value >0.05 as shown in Table no.1 and Graph no.1.

Probing pocket depth (PPD):

In Group 1, at baseline the mean probing pocket depth was 7.85±0.89 mm, reduced to 5.14±0.75 mm at 3rd month and 3.86±0.90 at 6th month. In Group 2, at baseline it was 7.67 ±0.80 mm, that reduced to 5.26±1.20 mm at 3rd month and 4.57±0.55 mm at 6th month as shown in Table no.2 and Graph no.2. In Group 1, the percentage (%) of PPD reduction was 34.52%, 50.82% at the end of 3rd and 6th month

respectively. In Group 2, it was 31.42% at 3rd month and 40.41% at 6th month as shown in Table no.5 and Graph no.5. On comparison between Group 1 and Group 2 from baseline to 3rd and 6th month, statistically it was highly significant with p-value <0.001.

Clinical attachment level (CAL):

In Group 1, the mean CAL at baseline was 8.71±0.94 mm, at the end of 3rd month the gain was 6.14±1.49 mm and at 6th month was 4.57±1.30 mm. In Group 2, the mean CAL at baseline was 8.28±0.60 mm, at the end of 3rd month was 6.83±1.32 mm and at 6th month was 5.82±1.12 mm as shown in Table no.3 and Graph no.3. In Group 1, the % of gain at 3rd month was 29.50% and at 6th month was 47.53%. In Group 2, at 3rd month it was 17.51% and at 6th month was 34.54% as shown in Table no.5 and Graph no.5. On comparison from baseline, the gain in CAL between the Groups at 3rd and 6th month time intervals were statistically highly significant with p-value <0.001.

Bone fill:

In Group 1, the defect at baseline was 7.57 ±0.75 mm. The bone fill was 6.33±1.11 mm and 4.42±0.53 mm at 3rd and 6th month respectively. In Group 2, the defect at baseline was 7.71 ±0.94 mm. The bone fill was 6.71±0.75 mm and 5.57±0.97 mm at 3rd and 6th month respectively as shown in Table no.4 and Graph no.4. The % of bone fill in Group 1 at baseline was 0%, at the end of 3rd month it was 16.38% and at the end of 6th month was 46.60%. In Group 2, at baseline it was 0 %, at 3rd month was 12.91% and at 6th month was 27.7% as shown in Table no.5 and Graph no.5. Comparing the mean defect fill between the Groups at 3rd and 6th month, statistically it was highly significant with p-value < 0.001.

TABLES

TABLE – 1

Comparison of mean plaque index scores, oral hygiene index scores, gingival index scores at baseline 3months and 6 months

PARAMETERS	BASELINE	3 MONTHS	6 MONTHS	p - value
Plaque index	1.14 ±0.68	0.80±0.52	0.51±0.42	>0.05*
Oral hygiene index	1.04±0.70	0.72±0.52	0.52±0.42	>0.05*
Gingival index	1.02±0.53	0.73±0.48	0.42±0.32	>0.05*

* p- value between baseline, 3months and 6months is >0.05 denotes not statistically significant at 5% level.

TABLE – 2

Inter group difference in mean probing pocket depth (PPD) at baseline 3months and 6 months

PROBING POCKET DEPTH	GROUP 1 (Mean±SD)	GROUP 2 (Mean±SD)	p – value
Baseline	7.85 ±0.89	7.67 ±0.80	< 0.001**
3 months	5.14 ±0.75	4.86±1.20	< 0.001**
6 months	3.86±0.90	4.57±0.55	< 0.001**

TABLE - 3

Inter group difference in mean clinical attachment level (CAL) at baseline

3months and 6 months

CLINICAL ATTACHMENT LEVEL	GROUP 1 (Mean±SD)	GROUP 2 (Mean±SD)	p – value
Baseline	8.71 ±0.94	8.28 ±0.60	< 0.001**
3 months	6.14±1.49	6.83±1.32	< 0.001**
6 months	4.57±1.30	5.42±1.12	< 0.001**

TABLE - 4

Inter group difference in mean bone fill at baseline 3months, and 6 months

DEFECT	GROUP 1 (Mean±SD)	GROUP 2 (Mean±SD)	P Value
Baseline	7.57 ±0.75	7.71 ±0.94	< 0.001**
3 months	6.33±1.11	6.71±0.75	< 0.001**
6 months	4.42±0.53	5.57±0.97	< 0.001**

** p- value between baseline, 3months and 6months is < 0.001 denotes statistically highly significant at 1% level

TABLE - 5

Inter group difference in percentage (%) of PPD reduction, CAL gain, and bone fill at baseline 3months and 6 months

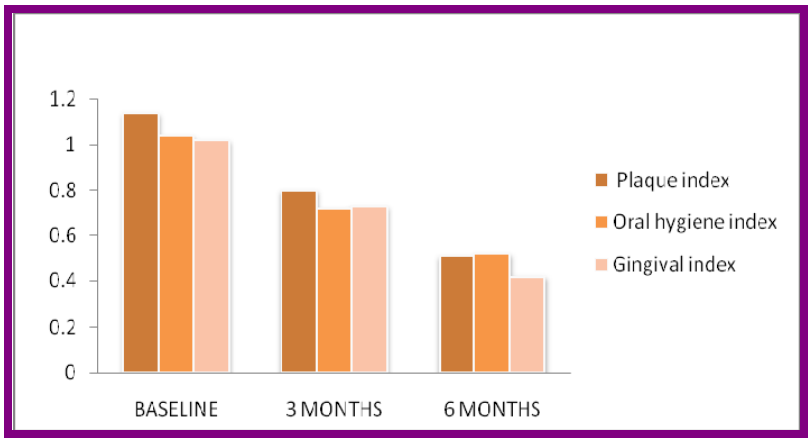
PARAMETERS	GROUP 1		GROUP 2		p-value
	3 months	6 months	3 months	6 months	
PPD reduction	34.52±0.14	50.82±0.15	31.42±0.5	40.41±0.31	<0.001**
CAL gain	21.58±0.40	47.53±0.38	17.51±0.86	34.54±0.40	<0.001**
Bone fill	16.38±0.48	46.60±0.29	12.91±0.19	27.7±0.03	<0.001**

** The overall comparisons were highly significant at 1% level of significance ($p<0.001$)

GRAPHS

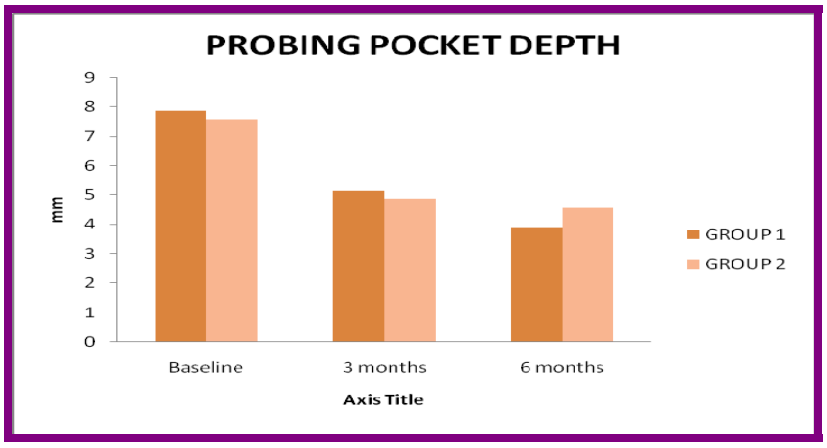
GRAPH-1

Comparison of mean changes in plaque index, gingival index, Oral hygiene index at baseline, 3months and 6 months



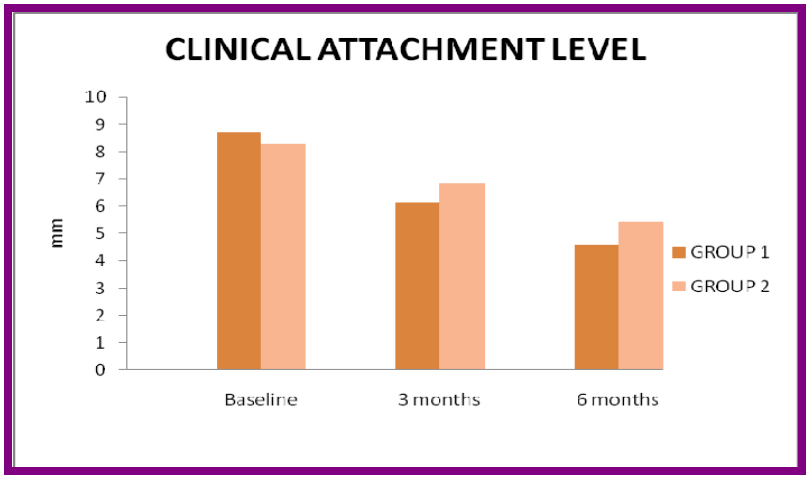
GRAPH-2

Comparison of mean changes in probing pocket depth (PPD) between the groups at baseline 3months and 6 months



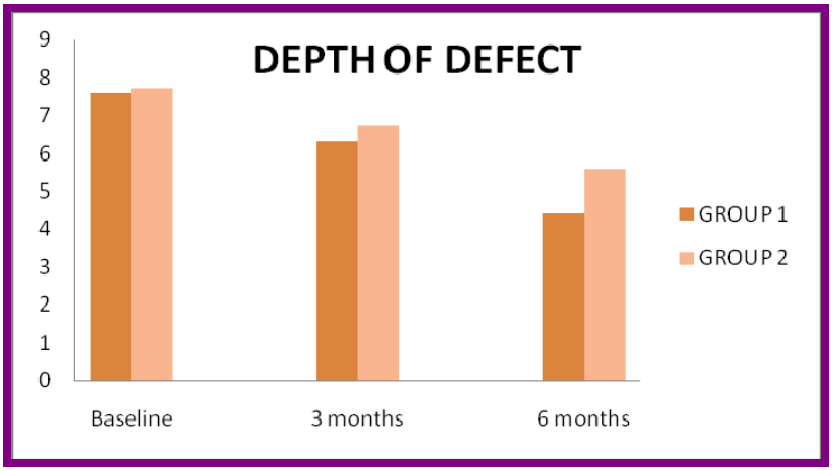
GRAPH-3

Comparison of mean changes in clinical attachment level (CAL) between the groups at baseline, 3months, and 6 months



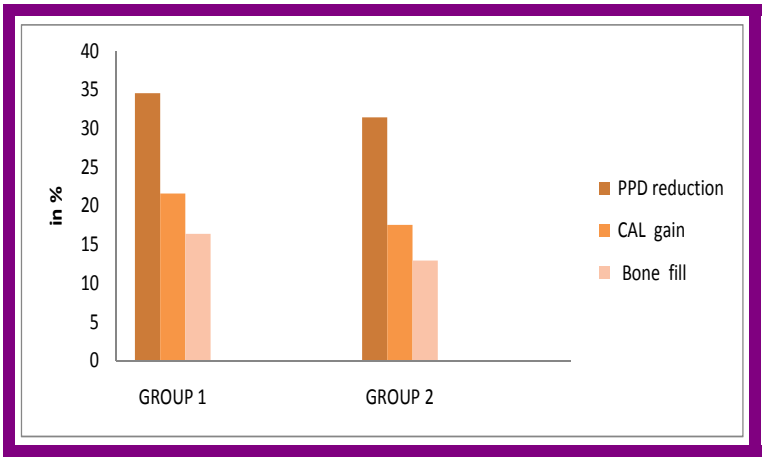
GRAPH -4

Comparsion of mean bone fill between the groups in baseline, 3 months and 6 months



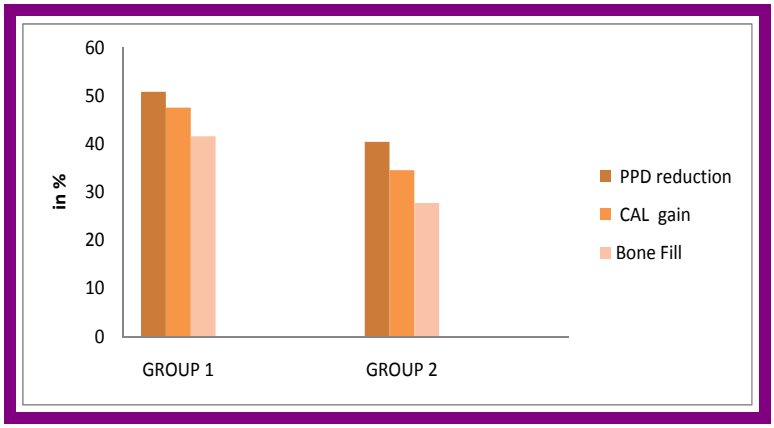
GRAPH-5

Comparsion between the groups in percentage of PPD reduction, CAL gain, bone fill at 3 months



GRAPH-6

Comparsion between the groups in percentage of probing depth reduction, attachment gain, bone fill at 6 months



Discussion

Periodontal disease is characterized by the presence of gingival inflammation, periodontal pocket formation, loss of connective tissue attachment and loss of alveolar bone around the affected teeth.[73] The treatment of these periodontal diseases by traditional methods may result in healing by the formation of long junctional epithelium.[3] Hence regenerative procedures have focused on the regeneration of new attachment apparatus including cementum, periodontal ligament and alveolar bone.[3,4] Autografts, allografts, xenografts and alloplasts are the most commonly used bone grafts in regenerative therapy.[7, 8]

Autogenous bone has been used with success and may be harvested from intra or extra-oral sites. Though the outcome of iliac grafts in many cases was very positive, complicated harvesting procedure, the need for hospitalization, donor site morbidity, potential ankylosis and root resorption were a few drawbacks.[74]

To overcome these drawbacks of extra-oral autografts, the present study utilizes intra-oral autogenous bone grafts obtained by Ebner grafter (Salvin Dental Specialists, USA) which is more advantageous as it eliminates the need for second surgical site, and reduces the post operative pain and swelling.[12]

In the present study autogenous bone graft was compared clinically and radiographically with demineralized freeze dried bone xenograft (Osseograft) combined with PRP in the treatment of human intrabony periodontal defects.

In the present study at baseline, **mean plaque index scores, oral hygiene index scores and gingival index scores** were found to be 1.14 ±0.68, 1.04±0.70 and 1.02±0.53 respectively. All these values reduced to 0.51±0.42, 0.52±0.42 and 0.42±0.32 respectively at the end of 6 months. The changes at different time intervals

(3^{rd} and 6^{th} months) was not statistically significant (p-value >0.05) but marked improvement in clinical parameters were observed. These results concur with the studies done by ***Gupta et al. (2007)*** [72] who observed marked improvement in the clinical parameters in terms of plaque index and gingival index after periodontal therapy. According to ***Gupta et al. (2007)*** [72] the patients undergoing periodontal therapy will maintain optimal oral hygiene and their compliance led to the improvement in their plaque index and gingival index scores.

The baseline **mean probing pocket depth (PPD)** in **Group 1** was 7.85±0.89 mm, and at the end of 6 months the mean PPD was 3.86±0.90 mm. And so the reduction in PPD was **3.99 mm** was obtained in **Group 1**. Similar results were reported by ***Halliday et al. (1969)*** [31] who observed probing pocket depth reduction of 4 mm following treatment of intrabony defects with intaoral autogenous bone grafts.

In **Group 2**, the mean probing pocket depth reduced from 7.67 ±0.80 mm to 4.57±0.55 mm at the end of 6 months. Thus the PPD reduction of **3.10 mm** was obtained in Group 2. This is in accordance with ***Hanna et al. (2004)*** [20] who showed that xenografts combined with PRP resulted in greater probing pocket depth reduction of 3.5 mm compared to xenografts alone.

In Group 1 the PPD reduction at the end of 6 months was **50.82%.** In Group 2 it was **40.41%**. The overall comparison between Group 1 and Group 2 were statistically highly significant with p-value <0.001. This is in agreement with ***Coverly, Toto et al. (1975)*** [34] who reported that the earlier occurrence of osteogenesis in the osseous coagulum grafted defects resulted in higher pocket depth reduction compared to xenografts. This rapid filling of the osseous defects may also serve to inhibit the

apical migration of the epithelial attachment during the early stages of repair, thereby inhibit the subsequent recurrence of the defect.[34]

In this study both the Groups 1 and 2 resulted in a significant attachment gain at the end of 6 months compared to baseline. In **Group 1,** the **mean gain in CAL** at the end of 6 months was **3.14 mm.** A study conducted by ***Halliday (1969)*** [31] reported that the treatment of intrabony defects with intraoral cancellous bone showed new attachment of 3.5 mm after 9 months.

In **Group 2,** the mean gain in CAL at the end of 6 months was **2.46 mm**. Similar result was observed by ***Xiang-ying et al. (2006)*** [66] who reported that the combined therapy of PRP and xenografts showed attachment gain of 1.67 mm. The higher gain compared to xenografts alone can be attributed to the addition of PRP which increases the migration and proliferation of the endothelial cells, periodontal ligament cells, pre-osteoblasts, osteoblasts and osteoclasts in the surgical site which stimulates bone regeneration.[78]

In Group 1 the gain at the end of 6 months was **47.53%**. In Group 2, at the end of 6 months was **34.54%**. There was 12% increase in gain in Group 1 compared to Group 2. On comparison between the Groups at different time intervals (3^{rd} and 6^{th} months) Group 1 was statistically highly significant with p-value <0.001. It is believed that autogenous bone grafts produce pronounced revascularization and enhances osteogenesis which resulted in marked gain in the clinical attachment level compared to other bone graft materials.[8]

In **Group 1**, the depth of the defect at baseline was 7.57 ±0.75 mm. At the end of 6 months it was reduced to 4.42±0.53 mm. In the present study, the **mean bone fill** at the 6th month was **3.15 mm**. The results concur with previous studies of ***Forum et al. (1975)*** [35] who reported the mean bone fill of 2.98 mm using bone blending. ***Hiatt and Schallhorn (1975)*** [37] also observed a mean bone fill of 3.5 mm using intra-oral cancellous bone in intrabony defects.

In **Group 2**, the depth of defect at baseline was 7.71 ±0.94 mm. At 6 months it was reduced to 5.57±0.97 mm. The **mean bone fill** at the 6th month was **2.14 mm**. Similar results were shown by ***Xiang-ying et al. (2006)*** [66] who reported a mean bone fill of 2.20 mm in intrabony defects grafted with xenografts and PRP.

In the present study, **Group 1** resulted in **46.60%** of **bone fill** at the end of 6th month. In **Group 2**, the bone fill was **27.7%** and at the end of 6th month. There was 19% increased bone fill in Group 1 compared to Group 2 which was statistically highly significant with p-value <0.001.

This is in accordance with ***Becker et al. (1998)*** [38] who observed that the healing of demineralized bone grafts was delayed due to the retention of non-vital bone particles within the host bone. In contrast, autografts heal with vital woven and lamellar bone with pronounced osteoclastic and osteoblastic activity. Also the demineralized bone grafts was primarily osteoconductive in nature and only act as space filler particles in the defect site whereas autogenous bone grafts heals by osteogenesis and retains viable osteoblasts.[6,8] In the early stages of healing of autografts new bone originates from the surviving osteoprogenitor cells and in later stages new bone originates from the osteoinduction response of the host bone.[36]

The present study shows overall reduction in the probing pocket depth, marked gain in the clinical attachment level and bone fill in both the groups which could be attributed to the resolution of tissue inflammation, reconstruction of the supporting periodontal structures in terms of alveolar bone, PDL which was in accordance with the previous studies by ***Carraro et al. (1975)*** [2]***, Hanna et al. (2004)*** [20]

Particle size of bone grafts plays an important role which results in greater bone fill, greater gain, and improved healing response in Group 1. According to ***Rivault et al. (1971)*** [32] the thin bone chips of osseous coagulum provides sooner and greater osteogenic activity than the thicker particles as found in allografts and xenografts.

In this study the osseous coagulum obtained using scraper also produces thin bone shavings which resulted in more regeneration compared to xenografts. According to ***Weinmann and Sicher (1969)*** [34] the smaller and thinner particles have the advantage of making them readily susceptible to hydrolyzing enzymes which dissolve their cementing substances and liberate minerals. These particles also results in rapid resorption which can lead to a local rise in calcium concentration and thus favour more bone formation.[10, 36, 76]

In our study Group 2 (xenograft and PRP) produced less PPD reduction, less CAL gain and decreased bone fill compared to Group 1 (autogenous bone grafts). The results corroborates the findings of previous studies done by ***Schlegel et al. (2004)*** [77] who have shown that PRP increases bone formation only in the initial stages of healing.

Nagata and Melo (2009) [78] also reported that the beneficial effect of PRP was limited to an initial healing period of 4 weeks. After 4 weeks the direct influence of PRP will fade away and physiological mechanisms of bone repair will continue to work according to the type of graft used. [78, 74]

Thus the regeneration of the periodontal attachment apparatus in the present study had a favorable clinical and radiological outcome using both bone replacement grafts. While comparing the 2 Groups, statistically highly significant difference ($p<0.001$) was noted in Group 1 regarding the clinical parameters and radiological parameters at 3rd and 6th months postoperatively.

However, limitations of this study include a small sample size and limited time frame for post surgical evaluation. In order to evaluate the true regenerative potential of the bone graft materials and biologic modifiers (PRP), a study design of larger sample size with longer follow-up period (more than 12 months) would be needed. Surgical reentry of treated sites would also provide credible data.

Summary And Conclusion

The present study involved a comparative clinical and radiographic evaluation of regenerative osseous surgery performed with autogenous bone grafts and demineralized xenograft (osseograft) combined with PRP. The study population comprised of 7 patients age ranging from 20-50 years. All patients returned for scheduled maintenance visits. A total of 14 intrabony defects were treated and post operative healing in the grafted areas was satisfactory. The following clinical parameters like plaque index, gingival index, oral hygiene index (simplified), probing pocket depth and clinical attachment levels were assessed at baseline, 3 months and 6 months. Hard tissue evaluation was done to measure the amount of bone fill in the grafted sites using Auto-CAD 2006 software.

Within the framework of this study, the following conclusions have been elucidated:-

1. Both Autogenous bone graft and combination of Demineralized Bovine derived Xenograft (Osseograft) with PRP, used as regenerative graft material in bone grafting yielded favorable clinical results in periodontal intrabony defects.
2. Probing pocket depth and gain in attachment level were significant in both Groups compared to their pre-operative levels.
3. Both the Groups exhibited significant amount of bone fill following therapy. But sites grafted with **autogenous bone grafts (Group 1)** showed a statistically **highly significant** ($p<0.001$) amount of **bone fill** indicative of better graft remodeling compared to sites grafted with mixture of xenograft and PRP (Group 2) at 3^{rd} and 6^{th} months post operatively.

4. Thus when mean scores were compared between Groups at 3rd and 6th months post- operatively, there was statistically highly significant difference ($p<0.001$) in Group 1 as far as clinical parameters and radiographic measurements were concerned.

The results presented here clearly demonstrate that the autogenous bone graft and combination of bovine derived xenograft (Osseograft) with PRP has the potential to promote predictable periodontal regeneration in the treatment of periodontal intraosseous defects. The results also indicate that surgical reconstructive treatment of intra-osseous defects with autogenous bone graft (Group 1) resulted in clinically and statistically significant higher probing pocket depth reduction, clinical attachment level gain and radiographic bone fill compared to xenograft mixed with PRP (Group 2).

However, it is necessary to have a large sample size and long term well controlled clinical trails to evaluate the true efficacy of these materials. Also further studies needed to be carried out to confirm the effects of PRP in periodontal regeneration.

ARMAMENTARIUM

BONE GRAFTING MATERIALS

Autogenous bone scraper

Xenograft

PREPARTION OF PRP

Collection of Blood sample

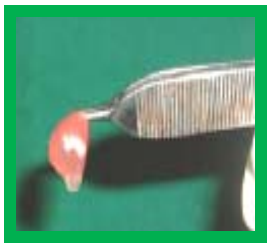
Table Top Centrifuge

Blood sample before centrifugation

After first centrifugation

PPP , PRP, and Red blood cells

PRP gel



Xenografts mixed with PRP

CLINICAL CASES

Group 1 – Autogenous bone grafts

Pre-operative view

Operative Procedure

Flap elevation

Autogenous bone graft

Autogenous bone graft placed

Sutures placed

Group 1

Probing pocket depth - Pre operative

6 months Post operative

Group 2 – Xenografts with PRP

Pre-operative view

Operative Procedure

Flap elevation

Mixture of Xenograft and PRP

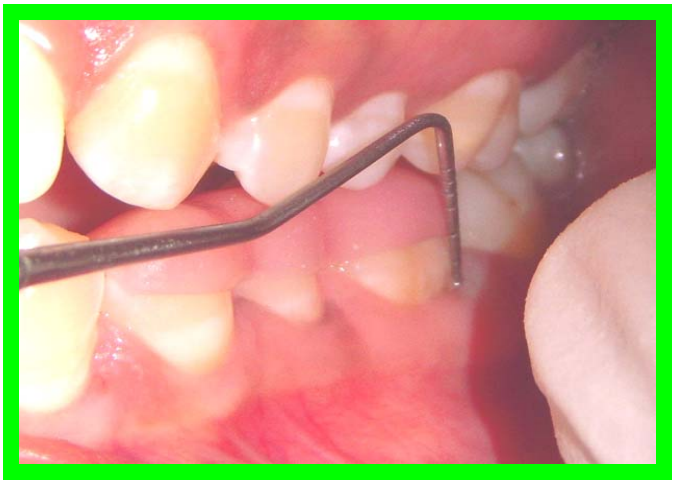
Bone graft placed

Sutures placed

Group 2

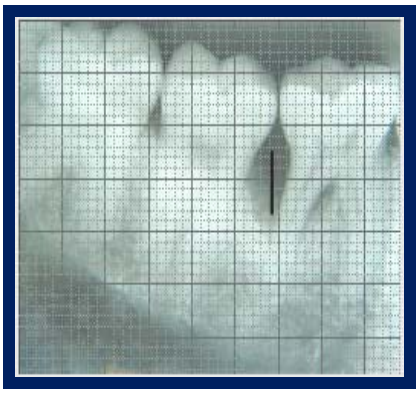
Probing pocket depth - Pre operative

6 months Post operative

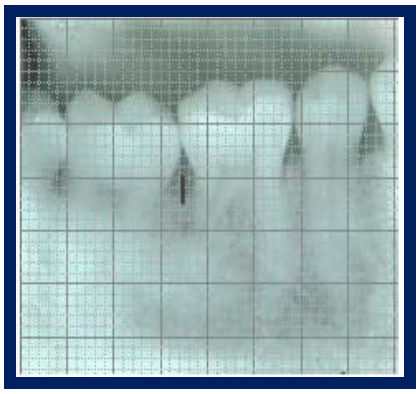


Group 1-Autogenous bone graft

Pre-op radiographic measurement

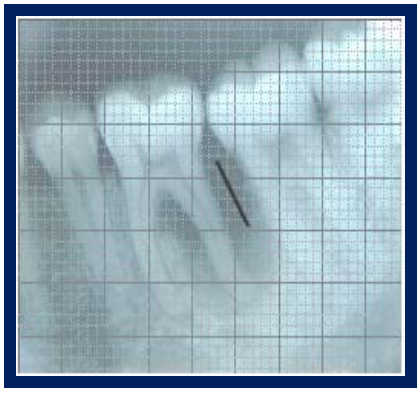


6 months Post-operative

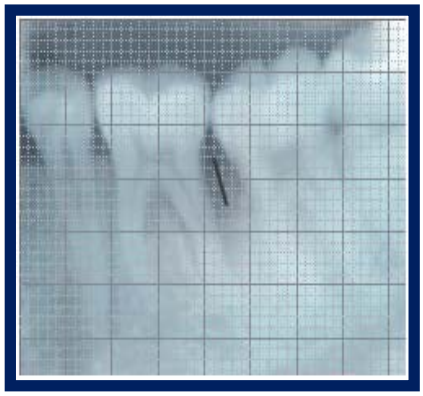


Group 2- Xenograft with PRP

Pre-op radiographic measurement



6 months Post-operative



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